Coal Workers' Lung Diseases

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Coal miners are subject to a number of lung diseases and disorders arising from their exposure to coal mine dust. These include pneumoconiosis, chronic bronchitis and obstructive lung disease. The occurrence and severity of disease depends on the intensity and duration of dust exposure. The specific composition of the coal mine dust also has a bearing on some health outcomes.

In the developed countries, where high prevalences of lung disease existed in the past, reductions in dust levels brought about by regulation have led to substantial drops in disease prevalence since the 1970s. In addition, major reductions in the mining work force in most of those countries over recent decades, partly brought about by changes in technology and resulting improvements in productivity, will result in further reductions in overall disease levels. Miners in other countries, where coal mining is a more recent phenomenon and dust controls are less aggressive, have not been so fortunate. This problem is exacerbated by the high cost of modern mining technology, forcing the employment of large numbers of workers, many of whom are at high risk of disease development.

In the following text, each disease or disorder is considered in turn. Those specific to coal mining, such as coal workers' pneumoconiosis are described in detail; the description of others, such as obstructive lung disease, is restricted to those aspects that relate to coal miners and dust exposure.

Coal Workers' Pneumoconiosis

Coal workers' pneumoconiosis (CWP) is the disease most commonly associated with coal mining. It is not a fast-developing disease, usually taking at least ten years to be manifested, and often much longer when exposures are low. In its initial stages it is an indicator of excessive lung dust retention, and may be associated with few symptoms or signs in itself. However, as it advances, it puts the miner at increasing risk of development of the much more serious progressive massive fibrosis (PMF).

Pathology

The classic lesion of CWP is the coal macule, a collection of dust and dust-laden macrophages around the periphery of the respiratory bronchioles. The macules contain minimal collagen and are thus usually not palpable. They are about 1 to 5 mm in size, and are frequently accompanied by an enlargement of the adjacent air spaces, termed *focal emphysema*. Though often very numerous, they are not usually evident on a chest radiograph.

Another lesion associated with CWP is the coal nodule. These larger lesions are palpable and contain a mixture of dust-laden macrophages, collagen and reticulin. The presence of coal nodules, with or without silicotic nodules (see below), indicates lung fibrosis, and is largely responsible for the opacities seen on chest radiographs. Macronodules (7 to 20 mm) in size may coalesce to form progressive massive fibrosis (see below), or PMF may develop from a single macronodule.

Silicotic nodules (described under *silicosis*) have been found in a significant minority of underground coal miners. For most, the cause may rest simply with the silica present in the coal dust, although exposure to pure silica in some jobs is certainly an important factor (e.g., among surface drillers, underground motormen and roof bolters).

Radiography

The most useful indicator of CWP in miners during life is obtained using the routine chest radiograph. Dust deposits and the nodular tissue reactions attenuate the x-ray beam and result in opacities on the film. The profusion of these opacities can be assessed systematically by using a standardized method of radiograph description such as that disseminated by the ILO and described else in this chapter. In this method, individual posterior-anterior films are compared to standard radiographs showing increasing profusion of small opacities, and the film classified into one of four major categories (0, 1, 2, 3) based on its similarity to the standard. A secondary classification is also made, depending on the reader's assessment of the film's similarity to adjacent ILO categories. Other aspects of the opacities, such as size, shape and region of occurrence in the lung are also noted. Some countries, such as China and Japan, have developed similar systems for systematic radiograph description or interpretation that are particularly suited to their own needs.

Traditionally, small *rounded* types of opacity have been associated with coal mining. However, more recent data indicate that *irregular* types can also result from exposure to coal mine dust. The opacities of CWP and silicosis are often indistinguishable on the radiograph. However, there is some evidence that larger sized opacities (type r) more often indicate silicosis.

It is important to note that a substantial amount of pathologic abnormality related to pneumoconiosis may be present in the lung before it can be detected on the routine chest x ray. This is particularly true for macular deposition, but it becomes progressively less true with greater profusion and size of nodules. Concomitant emphysema may also reduce the visibility of lesions on the chest x ray. Computerized tomography (CT)—particularly high-resolution computerized tomography (HRCT)—may permit visualization of abnormalities not clearly evident on routine chest x rays, although CT is not necessary for routine clinical diagnosis of miners' lung diseases and is not indicated for medical surveillance of miners.

Clinical aspects

The development of CWP, although a marker of excessive lung dust retention, in itself is often unaccompanied by any overt clinical signs. This should not, however, be taken to imply that the inhalation of coal mine dust is without risk, for it is now well known that other lung diseases can arise from dust exposure. Pulmonary hypertension is more often noted in miners who develop airflow obstruction in association with CWP. Moreover, once CWP has developed, it usually progresses unless dust exposure ceases, and may progress thereafter. It also puts the miner at greatly increased risk of development of the clinically ominous PMF, with the likelihood of subsequent impairment, disability and premature mortality.

Disease mechanisms

Development of the earliest change of CWP, the dust macule, represents the effects of dust deposition and accumulation. The subsequent stage, that is, the development of nodules, results from the lung's inflammatory and fibrotic reaction to the dust. In this, the roles of silica and non-silica dust have long been debated. On the one hand, silica dust is known to be considerably more toxic than coal dust. Yet, on the other hand, epidemiological studies have shown no strong evidence implicating silica exposure in CWP prevalence or incidence. Indeed, it seems that almost an inverse relationship exists, in that disease levels tend to be elevated where silica levels are lower (e.g., in areas where anthracite is mined). Recently, some understanding of this paradox has been gained through studies of particle characteristics. These studies indicate that not only the quantity of silica present in the dust (as measured conventionally using infrared spectrometry or x-ray diffraction), but also the bioavailability of the surface of the silica particles may be related to toxicity. For example, clay coating (occlusion) may play an important modifying role. Another important factor under current investigation concerns surface charge in the form of free radicals and the effects of "freshly fractured" versus "aged" silica-containing dusts.

Surveillance and epidemiology

The prevalence of CWP among underground miners varies with the kind of job, tenure and age. A recent study of US coal miners revealed that from 1970 to 1972 about 25 to 40% of working coal miners had category 1 or greater small rounded opacities after 30 or more years in mining. This prevalence reflects exposure to levels of 6 mg/m^3 or more of respirable dust among coal face workers prior to that time. The introduction of a dust limit of 3 mg/m^3 in 1969, with a reduction to 2 mg/m^3 in 1972 has led to a decline in disease prevalence to about half of the former levels. Declines related to dust control have been noted elsewhere, for example, in the United Kingdom and Australia. Unfortunately, these gains have been counterbalanced by temporal increases in prevalence elsewhere.

An exposure-response relationship for prevalence or incidence of CWP and dust exposure has been demonstrated in a number of studies. These have shown that the primary significant dust exposure variable is exposure to mixed mine dust. Intensive studies by British researchers failed to disclose any major influence of silica exposure, as long as the percentage of silica was less than about 5%. Coal rank (percentage carbon) is another important predictor of CWP development. Studies in the United States, the United Kingdom, Germany and elsewhere have given clear indications that the prevalence and incidence of CWP increases markedly with coal rank, these being substantially greater where anthracite (high rank) coal is mined. No other environmental variables have been found to exert any major effects on CWP development. Miner age appears to have some bearing on disease development, since older miners appear to be at increased risk. However, it is not entirely clear whether this implies that older miners are more susceptible, whether it is a residence time effect, or is simply an artefact (the age effect might reflect underestimation of exposure estimates for older miners, for example). Cigarette smoking does not appear to increase the risk of CWP development.

Research in which miners were followed-up with chest radiographs every five years shows that the risk of developing PMF over the five years is clearly related to the category of CWP as revealed on the initial chest x ray. Since the risk at category 2 is much greater than that at category 1, conventional wisdom at one time was that miners should be prevented from reaching category 2 if at all possible. However, in most mines there are usually many more miners with category 1 CWP compared to category 2. Thus, the lower risk for category 1 compared to category 2 is offset somewhat by the larger numbers of miners with category 1. On this showing, it has become clear that all pneumoconiosis should be prevented.

Mortality

Miners as a group have been observed to have increased risk of death from non-malignant respiratory diseases, and there is evidence that the mortality among miners with CWP is somewhat increased over those of similar age without the disease. However, the effect is smaller than the excess seen for miners with PMF (see below).

Prevention

The only protection against CWP is minimization of dust exposure. If possible, this should be achieved by dust suppression methods, such as ventilation and water sprays, rather than by respirator use or administrative controls, for example, worker rotation. In this respect, there is now good evidence that regulatory actions in some countries to reduce the level of dust, taken around the 1970s, has resulted in greatly reduced levels of disease. Transfer of workers with early signs of CWP to less dusty jobs is a prudent action, although there is little practical evidence that such programmes have succeeded in preventing disease progression. For this reason, dust suppression must remain the primary method of disease prevention.

Ongoing, aggressive monitoring of dust exposure and the conscious exertion of control efforts can be supplemented by health screening surveillance of miners. If miners are found to develop dust-related diseases, efforts at exposure control should be intensified throughout the workplace and miners with dust effects should be offered work in low-dust areas of the mine environment.

Treatment

Although several forms of treatment have been tried, including aluminium powder inhalation, and the administration of tetrandine, no treatment is known that effectively reverses or slows the fibrotic process in the lung. Currently, primarily in China, but elsewhere also, whole-lung lavage is being tried with the intent of reducing the total lung dust burden. Although the procedure can result in the removal of a considerable amount of dust, its risks, benefits and role in the management of miners' health are unclear.

In other respects, treatment should be directed towards preventing complications, maximizing the miners' functional status and alleviating their symptoms, whether due to CWP or to other, concomitant respiratory diseases. In general, miners who develop dust-induced lung diseases should evaluate their current dust exposures and utilize the resources of government and labour organizations to find the avenues available to reduce all adverse respiratory exposures. For miners who smoke, smoking cessation is an initial step in personal exposure management. Prevention of infectious complications of chronic lung disease with available pneumococcal and yearly influenza vaccines is suggested. Early investigation of symptoms of lung infection, with particular attention to mycobacterial disease, is also recommended. The treatments for acute bronchitis, bronchospasm and congestive heart failure among miners are similar to those for patients without dust-related disease.

Progressive Massive Fibrosis

PMF, sometimes referred to as complicated pneumoconiosis, is diagnosed when one or more large fibrotic lesions (whose definition depends on the mode of detection) are present in one or both lungs. As its name implies, PMF often becomes more severe over time, even in the absence of additional dust exposure. It can also develop after dust exposure has ceased, and may often cause disability and premature mortality.

Pathology

PMF lesions may be unilateral or bilateral, and are most often found in the upper or middle lobes of the lung. The lesions are formed of collagen, reticulin, coal mine dust and dust-laden macrophages, while the centre may contain a black liquid which cavitates on occasion. US pathology standards require the lesions to be 2 cm in size or larger to be identified as PMF entities in surgical or autopsy specimens.

Radiology

Large opacities >>1 cm) on the radiograph, coupled with a history of extensive coal mine dust exposure, are taken to imply the presence of PMF. However, it is important that other diseases such as lung cancer, tuberculosis and granulomas be considered. Large opacities are usually seen on a background of small opacities, but development of PMF from a category 0 profusion has been noted over a five-year period.

Clinical aspects

Diagnostic possibilities for each individual miner with large chest opacities must be appropriately evaluated. Clinically stable miners with bilateral lesions in the typical upper-lung distribution and with pre-existing simple CWP may present little diagnostic challenge. However, miners with progressive symptoms, risk factors for other disorders (e.g., tuberculosis), or atypical clinical features should undergo a thorough appropriate examination before the diagnostician attributes the lesions to PMF.

Dyspnoea and other respiratory symptoms often accompany PMF, but may not necessarily be due to the disease itself. Congestive heart failure (due to pulmonary hypertension and cor pulmonale) is a not infrequent complication.

Disease mechanisms

Despite extensive research, the actual cause of PMF development remains unclear. Over the years, various hypotheses have been proposed, but none is fully satisfactory. One prominent theory was that tuberculosis played a role. Indeed, tuberculosis is often present in miners with PMF, particularly in the developing countries. However, PMF has been found to develop in miners in whom there was no sign of tuberculosis, and tuberculin reactivity has not been found to be elevated in miners with pneumoconiosis. Despite investigation, consistent evidence of the role of the immune system in PMF development is lacking.

Surveillance and epidemiology

As with CWP, PMF levels have been declining in countries which have strict dust control regulations and programmes. A recent study of US miners revealed that about 2% of coal miners working underground had PMF after 30 or more years in mining (although this figure may have been biased by affected miners leaving the work force).

Exposure-response investigations of PMF have shown that exposure to coal mine dust, category of CWP, coal rank and age are the primary determinants of disease development. As with CWP, epidemiological studies have found no major effect of silica dust. Although it was thought at one time that PMF developed only on a background of the small opacities of CWP, recently this has been found not to be the case. Miners with an initial chest x ray showing category 0 CWP have been shown to develop PMF over five years, with the risk increasing with their cumulative dust exposure. Also, miners may develop PMF after cessation of dust exposure.

Mortality

PMF leads to premature mortality, the prognosis worsening with increasing stage of the disease. A recent study showed that miners with category C PMF had only one-fourth the rate of survival over 22 years compared to miners with no pneumoconiosis. This effect was manifested over all age groups.

Prevention

Avoidance of dust exposure is the only way to prevent PMF. Since the risk of its development increases sharply with increasing category of simple CWP, a strategy for secondary prevention of PMF is for miners to undergo periodic chest x rays and to terminate or reduce their exposure if simple CWP is detected. Although this approach appears valid and has been adopted in certain jurisdictions, its effectiveness has not been evaluated systematically.

Treatment

There is no known treatment for PMF. Medical care should be organized around ameliorating the condition and associated lung illnesses, while protecting against infectious complications. Although maintaining functional stability may be more difficult in patients with PMF, in other respects, management is similar to simple CWP.

Obstructive Lung Disease

There is now consistent and convincing evidence of a relationship between lung function loss and dust exposure. Various studies in different countries have looked at the influence of dust exposure on absolute values of, and temporal changes in, measurements of ventilatory function, such as forced expiratory volume in one second (FEV_1), forced vital capacity (FVC) and flow rates. All have found evidence that dust exposure leads to a reduction in lung function, and the results have been strikingly similar for several recent British and US investigations. These indicate that over the course of a year, dust exposure at the coal face brings about, on average, a reduction in lung function equivalent to smoking half a pack of cigarettes each day. The studies also demonstrate that effects vary, and a given miner may develop effects equal to, or worse than, those expected from cigarette smoking, particularly if the individual has experienced higher dust exposures.

The effects of dust exposure have been found in both those who have never smoked and in current smokers. Moreover, there is no evidence that smoking exacerbates the dust exposure effect. Rather, studies have generally shown a slightly smaller effect in current smokers, a result that may be due to healthy worker selection. It is important to note that the relationship between dust exposure and ventilatory decline appears to exist independently of pneumoconiosis. That is, it is not a requirement that pneumoconiosis be present for there to be reduced lung function. To the contrary, it appears rather that the inhaled dust can act along multiple pathways, leading to pneumoconiosis in some miners, to obstruction in others and to multiple outcomes in yet others. In contrast to miners with CWP alone, miners with respiratory symptoms have significantly lower lung function, after standardization for age, smoking, dust exposure and other factors.

Recent work on ventilatory function changes has involved the exploration of longitudinal changes. The results indicate that there may be a non-linear trend of decline over time in new miners, a high initial rate of loss being followed by a more moderate decline with continued exposure. Furthermore, there is evidence that miners who react to the dust may choose, if possible, to remove themselves from the heavier exposures.

Chronic Bronchitis

Respiratory symptoms, such as chronic cough and phlegm production, are a frequent consequence of work in coal mining, most studies showing an excess prevalence compared to non-exposed control groups. Moreover, the prevalence and incidence of respiratory symptoms has been shown to increase with cumulative dust exposure, after taking into account age and smoking. The presence of symptoms appears to be associated with a reduction in lung function over and above that due to dust exposure and other putative causes. This suggests that dust exposure may be instrumental in initiating certain disease processes that then progress regardless of further exposure. A relationship between bronchial gland size and dust exposure has been demonstrated pathologically, and it has been found that mortality from bronchitis and emphysema increases with increasing cumulative dust exposure.

Emphysema

Pathological studies have repeatedly found an excess of emphysema in coal miners compared to control groups. Moreover, the degree of emphysema has been found to be related both to the amount of dust in the lungs and to pathological assessments of pneumoconiosis. Furthermore, it is important to recognize that there is evidence that the presence of emphysema is related to dust exposure and to the percentage of predicted FEV_1. Hence, these results are consistent with the view that dust exposure can lead to disability through causing emphysema.

The form of emphysema most clearly associated with coal mining is focal emphysema. This consists of zones of enlarged air spaces, 1 to 2 mm in size, adjacent to dust macules surrounding the respiratory bronchioles. The current thinking is that the emphysema is formed from tissue destruction, rather than from distension or dilation. Apart from focal emphysema, there is evidence that centriacinar emphysema has an occupational origin, and that total emphysema, (i.e., the extent of all types) is correlated with tenure in mining, in those who have never smoked as well as in smokers. There is no evidence that smoking potentiates the dust exposure/emphysema relationship. However, there are indications of an inverse relationship between the silica content of lungs and the presence of emphysema.

The issue of emphysema has long been controversial, with some stating that selection bias and smoking make interpretation of pathological studies difficult. In addition, some consider that focal emphysema has only trivial effects on lung function. However, pathological studies undertaken since the 1980s have been responsive to earlier criticisms, and indicate that the effect of dust exposure may be more significant for miners' health than previously thought. This point of view is supported by recent findings that mortality from bronchitis and emphysema is related to cumulative dust exposure.

Silicosis

Silicosis, though associated more with industries other than coal mining, can occur in coal miners. In underground mines, it is found most frequently in workers in certain jobs where exposure to pure silica typically occurs. Such workers include roof bolters, who drill into the ceiling rock, which can often be sandstone or other rock with high silica content; motormen, drivers of rail transport who are exposed to the dust generated by sand placed on the tracks to lend traction; and rock drillers, who are involved in mine development. Rock drillers at surface coal mines have been shown to be at particular risk in the United States, with some developing acute silicosis after only a few years of exposure. Based on pathological evidence, as noted below, some degree of silicosis may afflict many more coal miners than just those working the jobs noted above.

Silicotic nodules in coal miners are similar in nature to those observed elsewhere, and consist of a whorled pattern of collagen and reticulin. One large autopsy study has revealed that about 13% of coal miners had silicotic nodules in their lungs. Although one job, (that of motorman) was notable for having a much higher prevalence of silicotic nodules (25%), there was little variation in the prevalence among miners in other jobs, suggesting that the silica in the mixed mine dust was responsible.

Silicosis cannot be reliably differentiated from coal workers' pneumoconiosis on a radiograph. However, there is some evidence that the larger type of small opacities (type r) are indicative of silicosis.

Rheumatoid Pneumoconiosis

Rheumatoid pneumoconiosis, one variant of which is called *Caplan's syndrome*, is the term used for a condition affecting dust-exposed workers who develop multiple large radiographic shadows. Pathologically, these lesions resemble rheumatoid nodules rather than PMF lesions, and often arise over a short time interval. Active arthritis or the presence of circulating rheumatoid factor are generally found, but occasionally are absent.

Lung Cancer

Included in the occupational exposures suffered by coal miners are a number of substances that are potential carcinogens. Some of these are silica and benzo(a)pyrenes. Yet, there is no clear evidence of an excess of deaths from lung cancer in coal miners. One obvious explanation for this is that coal miners are forbidden to smoke underground because of the danger of fires and explosions. However, the fact that no exposure-response relationship between lung cancer and dust exposure has been detected suggests that coal mine dust is not a major cause of lung cancer in the industry.

Regulatory Limits on Dust Exposure

The World Health Organization (WHO) has recommended a "tentative health-based exposure limit" for respirable coal mine dust (with less than 6% respirable quartz) ranging from 0.5 to 4 mg/m^3. WHO suggests a 2 in 1,000 risk of PMF over a working lifetime as a criterion, and recommends that mine-based environmental factors, including coal rank, percentage of quartz and particle size should be taken into account when setting limits.

Currently, among the major coal-producing countries, limits are based on regulating coal dust alone (e.g., 3.8 mg/m^3 in the United Kingdom, 5 mg/m^3 in Australia and Canada) or on regulating a mixture of coal and silica as in the United States (2 mg/m^3 when the per cent quartz is 5 or less, or (10 mg/m^3)/per cent SiO_2), or in Germany (4 mg/m^3 when the per cent quartz is 5 or less, or 0.15 mg/m^3 otherwise), or on regulating pure quartz (e.g., Poland, with a 0.05 mg/m^3 limit).

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